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Lipid peroxidation is a prerequisite for galactosamine-induced damage

Mykhailo Korda

The aim of the study was to investigate some mechanisms of D-galactosamine hepatotoxicity as well as the hepatoprotective effect of cresacin. Rats received 400 mg/kg D-galactosamine via intraperitoneal injection. Cresacin at a dose 20 mg/kg was administered intraperitoneally 10 min after D-galactosamine. The following parameters were measured: hepatic activities of superoxide dismutase, catalase, glutathione peroxidase, glutathione reductase and ceruloplasmin, intensity of liver lipid peroxidation and of mitochondrial and microsomal oxidative processes.

After intoxication the activation of free radical oxidation in the liver was observed along with attenuation of the antioxidant system, inhibition of oxygen absorption rate in mitochondria, uncoupling of oxidative phosphorylation and tissue respiration and depression of microsomal oxidation. Treatment of rats with cresacin prevented partially the toxic effects of D-galactosamine.

Chemiluminescence intensity was increased 1 h after intoxication while all other changes became apparent from 6 h after treatment. This fact suggests that lipid peroxidation may be a prerequisite for galactosamine-induced damage. The protection offered by cresacin was related to its capacity to inhibit lipid peroxidation.